Discerning aortic waves during intra-aortic balloon pumping and their relation to benefits of counterpulsation in humans

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Kolyva C, Pantalos GM, Giridharan GA, Pepper JR, Khir AW. Discerning aortic waves during intra-aortic balloon pumping and their relation to benefits of counterpulsation in humans. J Appl Physiol 107: 1497–1503, 2009. First published September 3, 2009; doi:10.1152/japplphysiol.00413.2009.—An explanation of the mechanisms leading to the beneficial hemodynamic effects of the intra-aortic balloon pump (IABP) is lacking. We hypothesized that inflation and deflation of the balloon would generate a compression (BCW) and an expansion (BEW) wave, respectively, which, when analyzed with wave intensity analysis, could be used to explain the hemodynamic benefits of IABP support. Simultaneous ascending aortic pressure (Pao) and flow rate (Qao) were recorded in 25 patients during control conditions and with IABP support of 1:1 and 1:2. Diastolic aortic pressure augmentation (Paug) and end-diastolic aortic pressure (ED Pao) were measured. The BCW and BEW were obtained by integrating the wave intensity pattern in the ascending aorta typically shows three characteristic waves. A forward compression wave (FCW) is generated by LV ejection at early systole and a FEW due to the LV wall contraction slowing down in late systole. Also, there is a BCW, caused by the reflection of the FCW at the periphery, that is observed in mid-systole. The BCW may not be seen in young normal subjects, as it varies in magnitude and arrival time at the ascending aorta according to factors such as vascular tone, age, and arterial compliance (10, 19). In addition to these waves originating from IABP function, additional mechanistic understanding of the hemodynamic benefits of IABP inflation and deflation might be achieved.

WIA is a time-domain method and can readily discriminate between forward-running waves that propagate in the same direction as mean blood flow and backward-running waves, traveling in the opposite direction (2, 18). These waves can be further classified as compression or expansion waves, according to whether they increase or decrease pressure, respectively. There are therefore four possible types of waves. A forward compression wave (FCW) increases the pressure and causes flow acceleration, while a backward compression wave (BCW) also increases the pressure but causes flow deceleration. Oppositely, a forward expansion wave (FEW) decreases pressure and causes flow deceleration, while a backward expansion wave (BEW) also decreases the pressure but causes flow acceleration (2, 18).

The wave intensity pattern in the ascending aorta typically shows three characteristic waves. A FCW is generated by LV rapid ejection at early systole and a FEW due to the LV wall contraction slowing down in late systole. Also, there is a BCW, due to the reflection of the FCW at the periphery, that is observed in mid-systole. The BCW may not be seen in young normal subjects, as it varies in magnitude and arrival time at the ascending aorta according to factors such as vascular tone, age, and arterial compliance (10, 19). In addition to these waves that are present in any normal subject, it is expected that there are four possible types of waves. A forward compression wave (FCW) increases the pressure and causes flow acceleration, while a backward compression wave (BCW) also increases the pressure but causes flow deceleration. Oppositely, a forward expansion wave (FEW) decreases pressure and causes flow deceleration, while a backward expansion wave (BEW) also decreases the pressure but causes flow acceleration (2, 18).

Although there are a great number of in vivo and in vitro reports in the literature investigating the beneficial effects of the IABP, a detailed study on the mechanism by which the IABP delivers these well-known hemodynamic benefits is still missing. Wave intensity analysis (WIA) could provide insight into this mechanism (2, 18). In the aorta, waves originate from cardiac contraction and relaxation. During IAB function additional waves are expected to be generated due to IAB inflation and deflation. WIA provides a way of identifying and quantifying these waves of various origins, and by focusing on the waves originating from IAB function, additional mechanistic understanding of the hemodynamic benefits of IAB inflation and deflation might be achieved.

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waves are anticipated to play a role in increasing pressure during inflation and decreasing pressure during deflation.

The aim of this study is to use WIA to explain mechanistically, in terms of wave travel, how the well-known beneficial hemodynamic effects of intra-aortic balloon counterpulsation are produced. We hypothesized that IAB inflation generates a BCW that leads to diastolic aortic pressure augmentation, while IAB deflation generates a BEW that causes reduction in end-diastolic aortic pressure. To explore this hypothesis we applied WIA to phasic simultaneous aortic pressure and velocity measurements and investigated whether the energies carried by the BCW and the BEW are related to aortic pressure parameters conventionally used to express inflation and deflation benefit, respectively.

METHODS

Study group. Patients supported by the IABP before surgery for ventricular assist device placement at the Jewish Hospital Heart and Lung Institute (Louisville, KY) were recruited for this investigation. The protocol was approved by the Human Subjects Protection Program Office of the University of Louisville, and written informed consent was obtained from all patients or their families.

The study population consisted of 25 patients (15 men). The mean age of the patients was 53 yr (SD 11). All patients had impaired LV function with ejection fraction < 20% and had been accepted for left ventricular assist device (LVAD) implantation either as a bridge to transplantation or as destination therapy. As their hemodynamic and end-organ function continued to deteriorate following selection for LVAD implantation, IABP support was instituted to stabilize the patients pending LVAD placement. Patients were supported with the IABP at least 2 days preoperatively, and IABP assistance was continued in the ICU for at least 1 day after LVAD implantation.

The patients were brought into the surgical suite with the IABP operating in the console automated mode or in a manual mode with the inflation and deflation timing set by a member of the perfusion staff. Patients underwent standard anesthetic induction followed by controlled ventilation with anesthesia maintenance using isoflurane.

Instrumentation and hemodynamic measurements. Intra-aortic balloons (7.5–9.5 F,Datascope) were inserted percutaneously via the femoral approach and were connected to the intra-aortic balloon pump (System 98 or CS100, Datascope). Balloon size varied between 34, 40, or 50 ml, according to patient height.

Following a midline incision and exposure of the heart, but before initiating cardiopulmonary bypass, blood pressure and flow measurements were obtained. A high-fidelity 5 F dual-pressure-sensor catheter (Millar Instruments) was used for obtaining simultaneous ascending aortic (Pao) and LV (Pv) pressure signals. The catheter was inserted through a purse-string suture in the ascending aorta and advanced retrograde through the aortic valve during transesophageal echocardiographic guidance, until the tip sensor was in the left ventricle. Flow (Qao) was measured at the same location as aortic pressure, with a perivascular flow probe (Transonic Systems) snugly fit around the ascending aorta. The aortic measurements were acquired close to the aortic root, just distal to the coronary ostia. After processing by corresponding pressure and flow consoles (Gould amplifier-model 13-6615-50, Gould Instruments and Transonic HT207 flowmeter) the hemodynamic signals and the ECG were recorded at 200 or 400 Hz for off-line analysis using a LabVIEW-based, digital data-acquisition system.

Protocol. In all patients Pao, Pv, Qao, and the ECG were recorded before LVAD placement with the IABP assisting every beat (1:1), every second beat (1:2) and also when the IABP was on the standby mode. As with usual clinical practice, the timing of IAB inflation and deflation was based on the aortic pressure signal recorded by the IABP using the fluid-filled catheter incorporated in the IAB. When the time limitations during the procedure allowed it, additional data were collected from a subset of patients, where the previous sets of measurements were repeated with the IAB timing based on the Pao signal. Under stable hemodynamic conditions data were collected continuously for ~15 s in each case, allowing 2 min between subsequent recordings for the hemodynamics to stabilize after an adjustment in assistance frequency.

Data analysis. Data processing was performed with custom software written in Matlab (version R2006a, The MathWorks). Aortic pressure and flow were smoothed using a Savitzky-Golay filter (22). Flow velocity (Uao) was derived from the flow measurements, using the nominal size of the flow probe as an indicator of the cross section of the aorta at the measuring location. Hardware-related time lags between the Pao and Uao signals were corrected based on the PU-loop, as has been previously described (11).

From the continuous recordings, a single representative beat was selected for each case (“1:1”: assisted beat of the 1:1 cycle; “1:2A”: nonassisted beat of the 1:2 cycle; “1:2A”: assisted beat of the 1:2 cycle; “off”: pump on standby mode). Averages per beat, as well as systolic and diastolic averages, were derived for Pao and Pv. Diastolic aortic pressure augmentation (PAUG) was determined by subtracting maximum systolic from maximum diastolic aortic pressure. End-diastolic aortic pressure was measured just before aortic valve opening. Stroke volume (SV) was defined as the area under the Qao curve during systole, and subsequently cardiac output (CO) was calculated as CO = SV·HR. The hydraulic work of the left ventricle (in J) was defined, similarly to other investigations, as (10, 27):

\[ \text{LVW} = \int_{0}^{T} P_{ao} Q_{ao} \, dt \]  

where \( T \) represents the duration of systole.

Net wave intensity normalized for the digitizing sampling rate (in W·m⁻²·s⁻²) was calculated from the time derivatives of the filtered signals as (5, 19, 21):

\[ \frac{dI}{dt} = \frac{dP_{ao}}{dt} \frac{dU_{ao}}{dt} \]  

The separated forward (\( dI_{+} \)) and backward (\( dI_{-} \)) wave intensities (in W·m⁻²·s⁻³) then followed from (5, 19):

\[ dI_{\pm} = \pm \frac{1}{4\rho c} \left( \frac{dP_{ao}}{dt} \pm \rho c \frac{dU_{ao}}{dt} \right)^2 \]

where \( \rho \) is blood density (1,040 kg/m³) and \( c \) is wave speed (in m/s). The product \( \rho c \) was determined from the PU-loop method as the slope of the linear part of the loop during early systole (11, 13). Following the usual convention, forward wave intensity represents waves traveling toward the periphery and backward wave intensity represents waves traveling toward the heart.

The dominant separated forward and backward waves were identified as the major peaks on the \( dI_{+} \) and \( dI_{-} \) profiles, respectively. These waves were further classified as compression and expansion waves, according to whether pressure was increasing or decreasing, respectively, at the time of the waves (19). The main separated waves were quantified by calculating the area under the peak, which represents the energy (in J·m⁻³·s⁻²) transported by the wave, per unit vessel cross-sectional area.

Statistical analysis. All statistical analysis was performed in SPSS (v 15.0). Data are expressed as means (SD). Comparisons between the different IABP assistance levels were performed with paired t-tests. According to the Bonferroni correction, to account for the multiple comparisons, statistical significance was assumed at \( P < 0.05/k \), where \( k \) is the number of multiple tests. Linear regression analysis was performed for investigating relations between continuous parameters. The slopes of regression lines corresponding to different assistance...
frequencies were compared using t-tests. Analysis of covariance was used to determine whether the pressure-measuring technique on which the timing of the IABP was based (fluid-filled or solid-state pressure catheter) was acting as a covariate in the regression lines between wave energy and pressure-derived parameters of IAB hemodynamic benefit.

RESULTS

Measurements. Acceptable flow measurements were not available in 15 patients due to problems with signal noise and distortion. Acceptable aortic pressure measurements were available in all patients, and LV pressure was obtained in 22 of the 25 patients.

For all 25 patients the measuring protocol was completed with timing of inflation and deflation based on the aortic pressure signal recorded at the tip of the IAB by the IAB fluid-filled catheter. In 12 of these patients the protocol was repeated with inflation and deflation timing based on the Pao signal.

Hemodynamic signals. Typical examples of left ventricular pressure and aortic root pressure and flow obtained from a 49-yr-old patient during 1:2 assistance are shown in the top panels of Fig. 1 for a nonassisted beat (Fig. 1A) and its preceding assisted beat (Fig. 1B).

The major hemodynamic benefits of IAB counterpulsation, namely diastolic aortic pressure augmentation and reduction in end-diastolic aortic pressure, are clearly noticeable in the aortic pressure signal of the assisted beat. The early-diastolic negative peak and the late-diastolic positive peak in aortic flow are induced by IAB inflation and deflation, respectively.

Hemodynamic parameters. The bar graphs in Fig. 2 summarize information regarding diastolic aortic pressure augmentation and reduction in end-diastolic aortic pressure in our patient group.

IAB inflation caused diastolic aortic pressure augmentation of 19.1 mmHg (SD 13.6) during the 1:2 cycle. Significantly higher Paug of 21.1 mmHg (SD 13.4) was achieved during the 1:1 sequence (P < 0.001).

End-diastolic aortic pressure decreased by 13.7% (P < 0.0001) during 1:1 assistance compared with standby conditions, from 50.9 mmHg (SD 15.1) to 43.9 mmHg (SD 15.7). A similar decrease was observed during 1:2 assistance.

Table 1 contains results on other hemodynamic changes observed during IAB counterpulsation. Mean aortic pressure increased both during 1:1 and 1:2 assistance compared with the standby state (P < 0.0001 for both). This increase was significantly higher with 1:2 support. Mean LV pressure decreased by 10.9% (P < 0.0001) with 1:1 counterpulsation, from 42.3 mmHg (SD 12.0) during standby to 37.7 mmHg (SD 12.0). During the 1:2 assistance, the benefit of IAB operation on mean LV pressure is evident on the beat following the assisted beat, and there was a significant decrease of 8.0% (P < 0.0001). Similar changes were observed in average systolic and diastolic LV pressure.

Cardiac output increased by 21.0% during 1:1 assistance compared with standby conditions, from 3.16 l/min (SD 1.10) to 3.82 mmHg (SD 1.43) (P < 0.001), but there was no such effect during 1:2 support. These changes took place without any changes in LV systolic hydraulic work. Heart rate and aortic wave speed remained constant throughout the measuring protocol.

Wave intensity pattern. Aortic root wave intensity obtained during 1:2 assistance is shown in the bottom panels of Fig. 1 for a nonassisted beat (Fig. 1A) and its preceding assisted beat (Fig. 1B). Three main waves generated by cardiac contraction and relaxation were always discernible in the wave intensity profile of the nonassisted beat (Fig. 1A). After the opening of the aortic valve a FCW appeared that was associated with a rapid increase in aortic pressure and flow. This was followed by a reflected BCW augmenting aortic pressure and opposing forward aortic flow. This sequence of waves was completed by a FEW, generated immediately before aortic valve closure and related to a decrease in both aortic pressure and flow. This wave intensity profile is comparable to previous in vivo re-
Table 1. Mean hemodynamic values at different assistance frequencies

<table>
<thead>
<tr>
<th>Assistance Frequency</th>
<th>1:2nA</th>
<th>1:2A</th>
<th>1:1</th>
<th>Off</th>
</tr>
</thead>
<tbody>
<tr>
<td>Praw, mmHg</td>
<td>58.9 (15.6)</td>
<td>70.4 (14.8)</td>
<td>66.5 (13.3)</td>
<td>61.7 (15.6)</td>
</tr>
<tr>
<td>Systolic</td>
<td>63.3 (16.4)</td>
<td>66.8 (15.9)</td>
<td>60.5 (15.1)</td>
<td>67.9 (16.2)</td>
</tr>
<tr>
<td>Diastolic</td>
<td>56.0 (15.3)</td>
<td>72.4 (14.6)</td>
<td>69.7 (12.9)</td>
<td>57.8 (15.4)</td>
</tr>
<tr>
<td>PTV, mmHg</td>
<td>38.9 (12.5)</td>
<td>41.2 (12.5)</td>
<td>37.7 (12.0)</td>
<td>42.3 (12.0)</td>
</tr>
<tr>
<td>Systolic</td>
<td>67.8 (16.6)</td>
<td>70.9 (16.8)</td>
<td>65.7 (16.6)</td>
<td>72.4 (17.2)</td>
</tr>
<tr>
<td>Diastolic</td>
<td>21.4 (9.4)</td>
<td>22.7 (10.7)</td>
<td>20.9 (9.9)</td>
<td>23.6 (10.2)</td>
</tr>
<tr>
<td>CO, l/min</td>
<td>3.85 (1.67)</td>
<td>3.25 (1.10)</td>
<td>3.82 (1.43)</td>
<td>3.16 (1.10)</td>
</tr>
<tr>
<td>HR, beats/min</td>
<td>93 (21)</td>
<td>91 (20)</td>
<td>92 (21)</td>
<td>94 (22)</td>
</tr>
<tr>
<td>LVW, J</td>
<td>0.45 (0.28)</td>
<td>0.42 (0.24)</td>
<td>0.44 (0.24)</td>
<td>0.39 (0.18)</td>
</tr>
<tr>
<td>c, m/s</td>
<td>6.0 (1.7)</td>
<td>6.9 (2.1)</td>
<td>5.9 (2.3)</td>
<td>7.0 (2.6)</td>
</tr>
</tbody>
</table>

Values are means (SD). Praw, average per beat aortic pressure; PTV, average per beat left ventricular pressure; CO, cardiac output; HR, heart rate; LVW, left ventricular systolic work; c, wave speed. Mean values for Praw and HR are based on 25 patients, for PTV on 22 and for CO, LVW and c on 10; “1:1,” assisted beat of the 1:1 cycle; “1:2nA,” nonassisted beat of the 1:2 cycle; “1:2A,” assisted beat of the 1:2 cycle; “Off,” intra-aortic balloon pump on standby mode. *P < 0.005 compared with off; †P < 0.001 compared with 1:1; ‡P < 0.0001 compared with 1:2nA.

Ports, although there are qualitative differences in the magnitude of the waves and the timing of the BCW (10, 19). The same three waves were present in the wave intensity contour of the assisted beat (Fig. 1B), but in addition, two pairs of waves caused by IAB inflation and deflation were also observed in all patients. Immediately after the closure of the aortic valve, a BCW accompanied by augmentation in early-diastolic aortic pressure and aortic backward flow was generated due to IAB inflation. The reflection of this wave at the closed aortic valve created in part the FCW that appeared immediately after the BCW and that was possibly further enhanced by the recoiling action of the aorta. IAB deflation induced a BEW that was accompanied by a steep decrease in end-diastolic aortic pressure and an increase in forward aortic flow. This wave was also reflected at the closed aortic valve as a FEW that appeared in the wave intensity profile immediately after the BEW.

Wave energy at different assistance frequencies. The mean energies over all patients of the two separated backward waves generated by IAB inflation and deflation are presented in Fig. 3, for the two different assistance cycles. The energy carried by the BCW was $1.1 \times 10^4 \text{J} \cdot \text{m}^{-2} \cdot \text{s}^{-2}$ (SD $0.8 \times 10^4$) at IABP assistance frequency 1:1, and it did not change considerably with assistance 1:2 ($P = 0.89$). The energy of the BEW was $1 \times 10^4 \text{J} \cdot \text{m}^{-2} \cdot \text{s}^{-2}$ (SD $0.5 \times 10^4$) at frequency 1:1, and, similarly to the BCW, there was no significant change in this energy during the 1:2 cycle ($P = 0.21$). Despite the 15% difference during 1:1 and the 28% difference during 1:2 assistance, there was no statistically significant difference in the energies carried by the BCW and the BEW at the same assistance frequency ($P = 0.39$ and $P = 0.10$, respectively). Due to the large variability in wave energy between patients, which is indicated by the large SDs, we emphasize that the results of the statistical analysis should be interpreted with caution.

Relation of balloon-generated waves with IAB hemodynamic benefit. The energy of the BCW generated by IAB inflation was correlated to diastolic aortic pressure augmentation (Fig. 4), with the lower wave energies corresponding to lesser hemodynamic benefit during inflation. Strong correlations were found for both assistance frequencies (1:1: $r = 0.83, P < 0.0001$; 1:2A: $r = 0.74, P < 0.01$), with no significant difference in slope between the two. Thus only the combined regression line for the two frequencies is presented in Fig. 4 ($r = 0.78, P < 0.0001$).

Figure 5 shows the relationship between the energy of the BEW generated by IAB deflation and end-diastolic aortic pressure. A significant negative correlation, with higher energy of BEW related to lower end-diastolic aortic pressure and therefore to higher hemodynamic benefit for the patient, was found for 1:1 assistance ($r = 0.78, P < 0.005$), as well as for 1:2 assistance ($r = 0.63, P < 0.05$). There was no significant difference between the slopes of these two lines, and therefore...
only the combined regression line for both assistance frequencies is presented in Fig. 5 \((r = 0.72, P < 0.0001)\).

**DISCUSSION**

In this study we applied WIA to pressure and flow measurements in the ascending aorta of humans during IAB counterpulsation. We examined the energy of the waves generated by balloon operation in relation to hemodynamic parameters conventionally used in the clinic for quantifying the benefit of IAB function. During IAB pumping, in all patients, a characteristic pattern of two pairs of forward and backward waves was evident. The energy of the BCW generated by IAB inflation correlated positively to Paug, whereas the energy of the BEW generated by IAB deflation correlated negatively to ED Pao.

**Hemodynamic effects.** It is well known that the net effects of IAB pumping on the systemic hemodynamics vary with factors such as arterial compliance, blood pressure, IAB volume, IAB positioning in the aorta, heart rate, and IAB timing of inflation and deflation \((3, 7, 14–17, 23, 24)\). Considering the large variations in the above factors within and between different study groups, the hemodynamic benefits observed in our patient population were in line with the findings of previous reports with minor deviations. We observed diastolic aortic pressure augmentation during IAB inflation and decrease in end-diastolic aortic pressure during IAB deflation. These are shown in Fig. 2 and are widely accepted as the main advantages of IAB counterpulsation \((7, 16)\). Due to the absence of coronary flow measurements, it is not possible to confirm whether diastolic coronary perfusion enhancement took place in our patient group during IAB inflation, although this is also a widely reported outcome in nonstenotic or moderately stenotic coronary vessels \((7–9, 12, 26)\). The decrease in LV afterload and subsequent decrease in metabolic demand that are typically associated with the reduction in end-diastolic pressure \((16)\) are implied in our patient group by the reduction in LV pressure (Table 1). The observed increase in cardiac output (Table 1) during counterpulsation is also in agreement with previously published data \((7, 16)\), although during 1:2 support this increase was not statistically significant. The increase in cardiac output, however, is not accompanied by a rise in LV systolic work. Therefore, it can be attributed either to an increase in the pumping efficiency of the LV per se or to the deflation of the IAB and the resulting afterload reduction, without the LV having to generate more work.

**Mechanisms.** As illustrated in Fig. 1, the ascending aortic wave intensity pattern of an assisted beat displays two distinct negative peaks that correspond to backward waves occurring during IAB inflation and deflation. Balloon inflation generates a BCW that causes an increase in aortic root pressure and backward flow. In contrast, balloon deflation generates a BEW that causes a decrease in aortic root pressure and an increase in forward flow. The hemodynamic effects of both backward IAB
compression and expansion waves are shown in the top panel of Fig. 1B.

The sudden increase in ascending aortic blood volume, due to the increased backward flow, and the consequent increase in aortic pressure during inflation is widely known as the primary benefit of IAB inflation (7, 16). Similarly, the rapid decrease in aortic volume, resulting in a reduction in LV afterload, is extensively reported as the principal hemodynamic benefit of deflation (7, 16). In this study we show that these hemodynamic effects have their origins in the BCW and BEW that occur during inflation and deflation, respectively. The causal relation between the waves and the hemodynamic effects is not proposed solely due to their relation in time. As shown in Fig. 4, the energy carried by the BCW is positively correlated to diastolic aortic pressure augmentation, which is commonly used as an index of inflation hemodynamic benefit. Higher energy of this wave and therefore a stronger push of the IAB toward the heart resulted in enhanced diastolic pressure augmentation. Similarly, in Fig. 5 it is illustrated that the energy carried by the BEW is negatively correlated to end-diastolic hemodynamic benefit; end-diastolic pressure was lower when the BEW carried more energy and therefore when the IABP could pull more. It is therefore possible to use the concept of wave generation and propagation in the ascending aorta to explain the primary mechanisms that lead to the hemodynamic benefits of IABP support.

**Methodological considerations.** It was possible in 12 patients to repeat the measurements with the IABP timed on the aortic root pressure signal, $P_{ao}$. This implies that these patients provided double the number of points for data and statistical analysis compared with the rest of the population. The possibility that some of the outcomes of this study might be biased, because patients who presented less complications in the OR and who were thus more suitable for a prolonged experiment contributed more to the data set, was investigated as follows. All $t$-tests for comparing mean values were repeated keeping the same number of points for each patient, by using only the fluid-filled catheter-based IABP timing measurements. There was no qualitative difference between the conclusions of this subset from those drawn from the complete data set. Further, it was established that using a fluid-filled or a solid state catheter to measure aortic pressure for IABP timing had no significant effect on the correlations between wave energy and hemodynamic parameters. There was no significant difference ($P = 0.655$) in the relationship between the energy of the BCW and $P_{aug}$ when using all measurements compared with using fluid-filled measurements only. Similarly, the correlation between the energy of the BEW and end-diastolic aortic pressure derived using all measurements was the same ($P = 0.134$) as the one corresponding to fluid-filled measurements only. Thus all available points were used for each patient for the regression lines presented in Figs. 4 and 5 for higher statistical power.

The theory proposed for the separation of the measured aortic pressure into windkessel pressure and wave pressure before applying WIA (25) could not be applied in the aorta during IABP support. The fundamental difficulty is that the aortic windkessel is discharging while the IAB is pumping, and as a result not only are the effects of the windkessel and the IAB in aortic pressure entirely convoluted and opposing each other, but also peripheral resistance is changing with time whereas the theory accounts for a constant resistance only.

It is evident from the large error bars in Fig. 3 that there is considerable variability in wave energy between patients, both for the BCW and the BEW. We speculate that this variability might have been induced by clinical factors that are also known to affect the hemodynamic effects of IAB pumping, such as arterial compliance, aortic pressure, and heart rate. However, the patient sample was small, and thus a more elaborate statistical analysis dividing the patients into subgroups could not be performed.

**Clinical implications.** Comparison of the hemodynamic benefits achieved with different assistance frequencies of the IABP shows that 1:1 support was superior to 1:2 in terms of diastolic pressure augmentation and increase in cardiac output and mean aortic flow, but 1:2 assistance was more efficient in increasing mean aortic pressure. End-diastolic aortic pressure, LV pressure, and LV systolic hydraulic work did not differ significantly between the two assistance frequencies. Although these observations were manifested in our patient group, a larger-scale study would be necessary before drawing general conclusions about a potential difference in support frequencies. Further, since the extent of the hemodynamic benefits and their clinical significance will vary with patient condition, conclusions drawn from this study are more applicable to intraoperative IABP patients requiring LVAD placement.

The energy carried by the BCW during IAB inflation was not significantly different from the energy carried by the BEW during deflation at corresponding assistance frequencies. Considering the energy carried by the waves as an index of the strength of the push and the pull of the IABP during inflation and deflation, respectively, this result suggests that the deflation phase of the IAB cycle is as important as the inflation phase in terms of IABP work, and its timing and resulting hemodynamic effect should not be overlooked in clinical practice.

**Conclusions.** The notion of wave generation and wave travel in the ascending aorta provides a mechanism to explain the beneficial hemodynamic effects of IAB inflation and deflation. IAB inflation generates a BCW, which has a pushing effect toward the heart, while IAB deflation generates a BEW, which has a pulling effect away from the heart. The energy of the IAB compression wave is correlated positively to diastolic aortic pressure augmentation, indicating that higher BCW energy leads to more augmentation. Also, the energy of the IAB expansion wave has a negative relationship with end-diastolic aortic pressure, indicating that higher BEW energy leads to lower end-diastolic pressure.

**GRANTS**

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**REFERENCES**


